Ischemic heart disease

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Introduction

- In > 90% of cases: the cause is: reduced coronary blood flow secondary to: obstructive atherosclerotic vascular disease

…so most of the time it is called: coronary artery disease

- Decades for the clinical syndromes to develop (may be since childhood)

- Causes other than coronary ischemia:

- ↑ demand (e.g., ↑ heart rate or HTN)
- Shock
- Anemia
- Pneumonia (↓ oxygenation)
- …etc.

Pathogenesis

…can affect any of the coronary arteries

-left anterior descending (LAD)

-left circumflex (LCX)

-right coronary artery (RCA)

…singly or in any combination

…clinically significant plaques can be located anywhere

…but tend to occur within the first several centimeters of the LAD and LCX, and along the entire length of the RCA

Pathogenesis, cont’d

- Fixed <70%: usually asymptomatic
- >70% = critical stenosis
- Fixed >90%...usually unstable angina (in the form of pain at rest)
- Thrombosis associated with a disrupted plaque often triggers the acute coronary syndromes (unstable angina-MI-SCD)
- Remember collateral perfusion

Pathogenesis, cont'd

…even partial luminal occlusion by thrombus can compromise blood flow sufficiently to cause a small infarction of the innermost zone of the myocardium (subendocardial infarct)

- Thrombosis as a process in atherosclerosis (other than that which is associated with rupture and sudden thrombosis) is associated with repair (organization and recanalization) and plaque enlargement

- Thrombosis may cause small emboli in intramyocardial branches with small microinfarcts

- Vasoconstriction is also important…it increases narrowing and increases rupture risk

…can be stimulated by:

(1) circulating adrenergic agonists

(2) locally released platelet contents

(3) imbalance between endothelial cell-relaxing factors (e.g., nitric oxide) and -contracting factors (e.g., endothelin) due to endothelial dysfunction

(4) mediators released from perivascular inflammatory cells

Pathogenesis, acute Plaque Change

- In most patients, unstable angina, infarction, and sudden cardiac death occur because of abrupt plaque change followed by thrombosis—hence the term acute coronary syndrome

…Rupture, fissuring, or ulceration

…by inducing thrombosis and hemorrhage into the core of plaques

can expand plaque volume

Vulnerable plaques (more likely to rupture)

- large atheromatous cores
- thin overlying fibrous caps
- lesions with a paucity of smooth muscle cells (more collagen) or large numbers of inflammatory cells (more metalloproteinases & collagen degradation)

- Unfortunately: two thirds of ruptured plaques are 50% stenotic or less before plaque rupture, and 85% exhibit initial stenotic occlusion of 70% or less

…it is impossible to predict plaque rupture in any given patient

Clinical syndromes

- Angina pectoris

… -stable

-unstable…due to superimposed thrombus (plaque rupture), embolus or spasm

-prinzmetal…typically but not necessarily at atherosclerotic plaques

- MI

- Heart failure

- Sudden cardiac death

Angina pectoris

= intermittent chest pain caused by transient, reversible myocardial ischemia

- ischemia-induced release of adenosine, bradykinin, and other molecules that stimulate the autonomic afferents…pain

- 3 types:

-Typical or stable angina

-Prinzmetal or variant angina…nitroglycerin and calcium channel blockers are good

-Unstable angina (also called crescendo angina)

Myocardial infarction

…commonly called: heart attack
…necrosis due to ischemia
…1/3 die and ½ before hospital
…10% before 40 years
…45% before age 65

- The vast majority of MIs are caused by acute coronary artery thrombosis

- In 10% of MIs:
  …transmural infarction occurs in the absence of occlusive atherosclerotic vascular disease
  -coronary artery vasospasm
  -embolization from mural thrombi (e.g., in the setting of atrial fibrillation) or valve vegetations

- Occasionally, subendocardial MI…thrombus/embolus may be absent

MI, cont’d

- An atheromatous plaque is suddenly disrupted by intraplaque hemorrhage or mechanical forces, exposing subendothelial collagen and necrotic plaque contents to the blood
- Platelets adhere, aggregate, and are activated, releasing thromboxane A2, adenosine diphosphate (ADP), and serotonin—causing further platelet aggregation and vasospasm
- Activation of coagulation by exposure of tissue factor and other mechanisms adds to the growing thrombus
- Within minutes, the thrombus can evolve to completely occlude the coronary artery lumen

MI and angiography

- Angiography performed within 4 hours of the onset of MI demonstrates coronary thrombosis in almost 90% of cases

- When angiography is performed 12 to 24 hours after onset of symptoms, however, evidence of thrombosis is seen in only 60% of patients…even without intervention

…so: early thrombolysis and/or angioplasty is effective

MI, sequence of events

-1-2 minutes after ischemia: loss of contractility…”stunned myocardium” for days even after reperfusion…may cause transient cardiac failure

-20-30 minutes: cell death

-2-3 hours: by EM

-6-12 hours: by LM

MI, vessels involved

- proximal left anterior descending (LAD) artery is the cause of 40% to 50% of all MIs

…results in infarction of:

-the anterior wall of the left ventricle

-the anterior two thirds of the ventricular septum

-most of the heart apex

…more distal occlusion of the same vessel may affect only the apex

- acute occlusion of the proximal left circumflex (LCX) artery (seen in 15% to 20% of MIs) will cause necrosis of the lateral left ventricle

- proximal right coronary artery (RCA) occlusion (30% to 40% of MIs) affects much of the right ventricle

MI, vessels involved, cont'd

- The posterior third of the septum and the posterior left ventricle are perfused by the posterior descending artery

  …The posterior descending artery can arise from either the RCA (in 90% of people) or the LCX

- The coronary artery—either RCA or LCX—that gives rise to the posterior descending artery and thereby perfuses the posterior third of the septum is considered the dominant vessel

  …right dominant heart VS left dominant

- Occasionally coronary occlusions are encountered in the left main coronary artery…widow maker

- Intramyocardial branches are rarely involved

- Remember collaterals between epicardial vessels

Patterns of MI

- Transmural…ECG changes

- Subendocardial

- Microscopic…small vessel occlusions
  -vasculitis
  -embolization of valve vegetations or mural thrombi
  -vessel spasm due to elevated catecholamines—either endogenous (e.g., pheochromocytoma or extreme stress), or exogenous (e.g., cocaine).

Lab investigations

- CK-MB
- Troponin I and troponin T

Complications

- Contractile dysfunction

…cardiogenic shock occurs in roughly 10% of patients with transmural MIs and typically is associated with infarcts that damage 40% or more of the left ventricle

- Papillary muscle dysfunction

…postinfarct mitral regurgitation

- Myocardial rupture

…1-5%

…3-7 days

…age older than 60 years, anterior or lateral wall infarctions, female gender, lack of left ventricular hypertrophy, and first MI (as scarring associated with prior MIs tends to limit the risk of myocardial tearing)

Complications, con’d

- Arryhythmias:

…90%

…in STEMI more than non-STEMI

… The risk of serious arrhythmias (e.g., ventricular fibrillation) is greatest in the first hour and declines thereafter

- Pericarditis
- Chamber dilation
- Ventricular aneurysm
- Chronic ischemic heart disease

Differences

- Large transmural infarcts are associated with a higher probability of cardiogenic shock, arrhythmias, and late CHF
- Patients with anterior transmural MIs are at greatest risk for free wall rupture, expansion, formation of mural thrombi, and aneurysm formation
- Posterior transmural infarcts are more likely to be complicated by serious conduction blocks, right ventricular involvement, or both
- Patients with anterior infarcts have a much worse clinical course than those with posterior infarcts
- Subendocardial infarcts, thrombi may form on the endocardial surface, but pericarditis, rupture, and aneurysms rarely occur

Thank You